All-Cause and Cause-Specific Mortality by Socioeconomic Status Among Employed Persons in 27 US States, 1984–1997

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Objectives. We investigated mortality differences according to socioeconomic status (SES) for employed persons in 27 states during 1984–1997.

Methods. SES was determined for persons aged 35–64 years according to the "usual occupation" listed on their death certificates. We used US Census denominator data.

Results. For all-cause mortality, rate ratios from lowest to highest SES quartile for men and women were 2.02, 1.69, 1.25, and 1.00 and 1.29, 1.01, 1.07, and 1.00, respectively. Percentage of all deaths attributable to being in the lowest 3 SES quartiles was 27%. Inverse SES gradients were strong for most major causes of death except breast cancer and colorectal cancer. Heart disease mortality for highest and lowest SES quartiles dropped 45% and 25%, respectively, between 1984 and 1997.

Conclusions. Mortality differences by SES were sustained through the 1990s and are increasing for men. (*Am J Public Health.* 2004;94:1037–1042)

Several large cohort studies in the United States have shown that mortality rates are higher among those of low versus high socioeconomic status (SES). Higher levels of standard risk factors for those with low SES do not appear to entirely account for this fact.[1–5]

Several studies have also considered temporal changes in mortality by SES and have shown that cardiovascular mortality has been decreasing faster for higher-SES groups from the 1950s to the 1980s.[6–10] Few data on SES and mortality have been published in recent years. The only study to have yielded more recent data was based on the American Cancer Society population, which has a higher SES than the general population. We examined whether previously observed SES differences in mortality persisted during the 1990s and how these differences changed over time in a population reasonably representative of the US population: employed persons aged 35–64 years in 27 states during the period 1984–1997.

METHODS

Numerator data for mortality rates consisted of all deaths and deaths from major specific causes per the *International Classification of Diseases, Ninth Revision* (*ICD-9*)[11]: coronary heart disease (CHD) (*ICD-9* codes 410–414), stroke (*ICD-9* codes 430–438), diabetes (*ICD-9* code 250), chronic obstructive pulmonary disease (COPD) (*ICD-9* codes 490–492, 496), all cancer (*ICD-9* codes 140–208), lung cancer (*ICD-9* code 162), breast cancer (*ICD-9* code 174), colorectal cancer (*ICD-9* codes 152–154), and external causes (*ICD-9* codes E800–E978). A death was included if the death certificate listed usual occupation and occurred in a decedent aged 35–64 years during the period 1984–1997 in a state that participated in the National Occupational Mortality Surveillance (NOMS) program. The NOMS data were derived from 27 states that, during the period 1984–1997, coded usual occupation and industry on their death certificates with US Census codes[12,13]; the program was coordinated by the National Institute for Occupational Safety and Health. These states were Arkansas, Colorado, Georgia, Hawaii, Indiana, Idaho, Kansas, Kentucky, Maine, Missouri, Nebraska, Nevada, New Hampshire, New Jersey, New Mexico, North Carolina, Ohio, Oklahoma, Pennsylvania, Rhode Island, South Carolina, Tennessee, Utah, Vermont, Washington, West Virginia, and Wisconsin; however, not every state participated each year. Data from an average of 19 states and 8 million people were included each year.

Deaths in which usual occupation was not reported (2%) and deaths of retired persons, military personnel, housewives, students, volunteers, and the unemployed (20% of all deaths, 7% for men and 43% for women) were excluded. Female homemakers (typically listed as "housewife" on the death certificate) accounted for most of the exclusions. The same exclusions were made in the denominator data. Deaths were stratified by age (in groups of 5 years), race (White or Black), and gender. Numerator (and denominator) data from races other than White or Black represented less than 1% of the data and were excluded.

Denominator data for mortality rates consisted of occupation-specific populations for all persons aged 35–64 years who were currently employed or who had been employed within the past 5 years and is available from the US Census Bureau for each NOMS-participating state (Carolyn Carbaugh; Housing and Household Economic Statistics Division, US Census Bureau; written communication; 2000). Population data, like numerator data, were stratified by age, race, and gender. Data for the period 1984–1990 were derived from interpolation between the 1980 and 1990 censuses; those for the period 1991–1997 were derived from the monthly current population surveys conducted by the US Census Bureau.[14]

The fact that the numerator consisted of deaths with "usual occupation" but the denominator consisted of the population currently employed meant that a one-to-one correspondence did not exist between numerator and denominator. Kuntz et al.[15] have discussed this problem in European SES studies; however, this problem is not critical in our data. Both numerator and denominator used the same coding scheme (US Census codes) to classify occupation. Two studies[16,17] have shown that for subjects of working age, "usual occupation" corresponded to "current occupation" 70%–80% of the time. Because we grouped both numerator and denominator data into 4 equal-sized SES groups, it is reasonable to assume that numerator and denominator data corresponded well within these broad groups.

We divided deaths (numerator) and populations (denominator) into socioeconomic groups according to Nam–Powers scores[18] for occupation. These scores are rankings (1 through 99) based on 1980 and 1990 census data on income and educational level by occupation and are assigned for each occupation that has a US Census code.[12,13] A list of occupations and their corresponding scores was made available to us by E. Terrie, PhD (written communication, 1996). Some example occupations and their corresponding Nam–Powers scores were doctor (99 [the highest possible score]), epidemiologist (93), elementary-school teacher (83), insurance adjuster (67), car salesman (58), secretary (51), plumber (50), carpenter (40), bartender (34), waiter (23), cashier (15), and maid (8). We grouped all occupation-specific deaths and populations into gender-specific quartiles according to their score (cutpoints: men=40, 63, 81; women=29, 49, 75).

We determined mortality rates for socioeconomic quartiles by dividing age-specific (5-year categories), year-specific, gender-specific, and race-specific numerator data by denominator data. Analyses were conducted by Poisson regression with SAS.[19] Models analyzing mortality by SES were adjusted for age (in groups of 5 years), race (White or Black), gender, and calendar time (either categorical or continuous). Variances of rate ratios were corrected for overdispersion. Trends over time for different SES quartiles were estimated with the coefficient of the variable for calendar year (continuous) in separate models for each SES quartile; analyses used the formula $1 - \exp(\beta)$ when the coefficient was negative (rates falling) and $\exp(\beta)-1$ when the coefficient was positive (rates increasing), with adjustments for other covariates (age, race, gender).

To evaluate possible confounding by state owing to nonconstant state participation from year to year, we analyzed data for a set of 10 NOMS states that were consistently available for the years 1984 through 1996. Results were virtually identical to those using the complete data set. We also tested variables for region of residence (groupings of states); these variables had little impact on SES effects.

We evaluated possible interactions between race and SES in our statistical model by testing the statistical significance (at the .05 level) of a race-by-SES interaction term (treated as a continuous variable). To assess smoking habits by SES, gender, and race in the US population, we obtained data from a large national survey, the third National Health and Nutrition Examination Survey (NHANES III), via a public-use data set.[20] Through use of appropriate sampling weights, we derived gender- and race-specific proportions of current smokers by quartiles of a poverty index (a variable on the NHANES III data set) among persons aged 20–64 years.

RESULTS

Table 1 presents the deaths and person-time data by SES quartile. Table 2 displays SES results for all causes combined, as well as all specific causes. For men, all outcomes showed consistent trends of increasing mortality with decreasing SES (after control for age, race, and calendar time). The lowest SES group generally had 2 to 3 times the death rate of the highest SES group for all causes and for each specific cause except colorectal cancer, which showed only a weak inverse SES gradient. The inverse SES gradients were most marked for COPD and external causes.

Women also generally showed inverse SES gradients that were weaker than those showed by men. The strongest inverse gradients were seen for CHD, diabetes, and COPD. In contrast, a strong positive SES gradient was seen for breast cancer and a weak gradient was observed for colorectal cancer.

Table 3 shows trends over time within each SES group for the period 1984–1997. Well-known changes in mortality rates for the total population in recent years are reflected in this table—for example, the decrease in CHD mortality rates and the increase in diabetes mortality rates. The most notable differences in trends over time by SES quartile were seen in men, for whom SES differences

TABLE 1—Number, by SES Quartile,[a] of Deaths[b] and Person-Years[c] for Employed US Adults Aged 35–64 Years, 1984–1987

	All Deaths	CHD	Stroke	Diabetes	COPD	External Causes	Lung Cancer	Breast Cancer	Colorectal Cancer	Person-Years / 10^6
SES 1 (highest)	206 320	38 276	6866	4290	3469	48 240	20 929	10 368	8157	56.8
SES 2	266 320	54 518	9226	6105	5824	40 098	29 379	9517	9934	57.0
SES 3	341 182	68 263	12 664	7736	10 012	30 659	40 949	10 977	10 134	58.6
SES 4 (lowest)	517 124	97 817	22 173	13 527	14 925	24 194	55 370	11 298	12 793	60.4
Total	1 330 886	258 874	50 929	32 658	34 230	143 191	146 567	42 160	40 478	232.9
Rate $\times 10^5$ for total population	571	111	21	14	15	61	63	18	17	

Note. CHD = coronary heart disease; COPD = chronic obstructive pulmonary disease; SES = socioeconomic status quartile.
[a]SES quartile is defined by occupation and corresponding Nam–Powers scores (based on income and education); the 4 gender-specific quartiles have Nam–Powers scores of < 40, 40–62, 63–80, and ≥ 81 for men and < 29, 29–48, 49–74, and ≥ 75 for women.
[b]Deaths are all deaths of persons aged 35–64 years; data are from death certificates with usual occupation coded, excluding housewives, military, students, the retired, or volunteers.
[c]Person-years are based on yearly counts of all persons aged 35–64 years who reported a current occupation or an occupation within the past 5 years.

TABLE 2—All-Cause and Cause-Specific Mortality Rate Ratios and 95% Confidence Intervals: 1984–1997

	All Causes	Coronary Heart Disease	Stroke	Diabetes	COPD	External Causes	Lung Cancer	Breast Cancer	Colorectal Cancer
Men vs women[a]	2.27 (2.22–2.32)	4.22 (4.11–4.34)	1.53 (1.49–1.58)	1.69 (1.64–1.75)	2.03 (1.96–2.11)	3.42 (3.33–3.52)	2.29 (2.22–2.35)	NA	1.48 (1.44–1.52)
Blacks vs Whites[a]	2.16 (2.10–2.22)	1.51 (1.47–1.56)	3.88 (3.76–4.00)	3.01 (2.90–3.14)	0.97 (0.91–1.03)	1.79 (1.65–1.75)	1.62 (1.56–1.68)	1.77 (1.70–1.83)	1.76 (1.69–1.82)
SES quartile, men[b]									
1 (lowest)	2.02 (1.95–2.09)	1.88 (1.83–1.93)	2.25 (2.14–2.37)	2.19 (2.07–2.32)	3.59 (3.35–3.83)	2.67 (2.58–2.78)	2.15 (2.07–2.23)	NA	1.21 (1.16–1.27)
2	1.69 (1.63–1.74)	1.68 (1.63–1.73)	1.79 (1.70–1.90)	1.85 (1.74–1.97)	3.10 (2.89–3.32)	2.12 (2.04–2.20)	2.03 (1.96–2.11)	NA	1.17 (1.12–1.23)
3	1.25 (1.21–1.29)	1.28 (1.24–1.32)	1.26 (1.19–1.33)	1.39 (1.30–1.48)	1.58 (1.46–1.70)	1.38 (1.33–1.44)	1.34 (1.28–1.42)	NA	1.12 (1.07–1.17)
4 (highest)	1.00	1.00	1.00	1.00	1.00	1.00	1.00	NA	1.00
SES quartile, women[b]									
1 (lowest)	1.29 (1.25–1.32)	1.84 (1.76–1.93)	1.53 (1.44–1.62)	1.85 (1.72–2.00)	2.09 (1.91–2.30)	1.41 (1.35–1.48)	1.31 (1.25–1.37)	0.76 (0.73–0.79)	0.91 (0.86–0.96)
2	1.01 (0.97–1.04)	1.25 (1.21–1.32)	1.11 (1.05–1.18)	1.13 (1.05–1.23)	1.43 (1.30–1.58)	0.97 (0.93–1.02)	1.04 (1.00–1.10)	0.80 (0.77–0.84)	0.89 (0.84–0.94)
3	1.07 (1.04–1.11)	1.29 (1.22–1.37)	1.17 (1.11–1.25)	1.24 (1.14–1.35)	1.35 (1.22–1.25)	1.10 (1.05–1.16)	1.11 (1.05–1.16)	0.90 (0.86–0.94)	0.96 (0.90–1.02)
4 (highest)	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00

Note. COPD = chronic obstructive pulmonary disease; SES = socioeconomic status; NA = not available.
[a]Model included terms for age, calendar year, gender, race, and SES.
[b]Model was restricted to either men or women and included terms for age, calendar year, race, and SES.

TABLE 3—Annual Percentage Change[a] in Mortality Rates for Total Population and by SES Quartile: 1984–1997

	All Causes	CHD	Stroke	Diabetes	COPD	External Causes	Lung Cancer	Breast Cancer	Colorectal Cancer
Total population	−0.6	−3.3	−1.3	4.0	0.0	−0.5	−0.8	−1.6	−1.1
SES quartile, men									
1 (lowest)	−0.4	−2.6	−1.1	4.2	0.1	−0.6	−0.4	NA	0.1
2	−1.0	−3.5	−1.2	4.3	−1.3	−0.3	−1.8	NA	−0.4
3	−1.7	−4.7	−2.2	3.2	−1.5	−1.4	−2.4	NA	0.4
4 (highest)	−1.9	−4.9	−2.0	2.7	−1.7	−1.6	−3.6	NA	−1.9
SES quartile, women									
1 (lowest)	0.2	−2.0	−1.8	4.2	3.9	0.1	1.8	−1.1	−1.2
2	−0.3	−2.5	−0.1	4.0	2.7	0.2	1.1	−2.3	−2.6
3	−0.9	−3.4	−1.4	1.8	0.0	−1.7	0.3	−1.6	−1.6
4 (highest)	0.2	−2.2	−1.1	6.2	1.5	−0.5	0.3	−1.3	−1.3

Note. CHD = coronary heart disease; COPD = chronic obstructive pulmonary disease; SES = socioeconomic status quartile; NA = not available.
[a]Annual percentage change was estimated via the coefficient for calendar time (continuous), using the formula 1−exp(β) when the coefficient was negative (rates falling), exp(β)−1 when the coefficient was positive (rates increasing), in a model which that adjusted for other covariates. Separate models were run for each gender/SES group.

increased over time for all causes examined. The strongest of the increasing SES differences over time were for CHD, COPD, lung cancer, and diabetes; similar but weaker patterns were seen for stroke, colorectal cancer, and external-cause mortality. For women, consistent differences in time trends by SES were seen only in lung cancer, for which rates increased in all SES groups (the opposite from the observed trend for men) and more rapidly for lower-SES groups.

In separate analyses of in-hospital (31%) and out-of-hospital (69%) CHD deaths in men and women combined, the annual decrease in mortality over the 14-year period was 4.6% for in-hospital deaths versus 2.7% for out-of-hospital deaths. The percentage annual decreases in in-hospital deaths were 3.5, 3.9, 6.1, and 6.2 by ascending SES quartiles; the corresponding figures were 1.9, 2.1, 3.9, and 3.8 for out-of-hospital deaths, by ascending SES quartiles.

Gender-specific statistical tests of interaction between the SES gradient and race were significant at the .05 level for 12 of 17 gender–disease combinations tested. Although in many instances SES gradients were similar for Blacks and Whites, the large sample size resulted in statistical significance. For all-cause mortality, Black men had a slightly steeper inverse SES gradient (rate ratios: 2.43, 1.65, 1.26, 1.00, by ascending SES) than did White men (rate ratios: 1.95, 1.71, 1.25, 1.00); there were no apparent differences between Black and White women. Black men tended to show a steeper inverse SES gradient than did White men across all specific causes. Differences between Black and White men were greatest for stroke (rate ratios: Blacks=3.10, 2.05, 1.38, 1.00; Whites=2.10, 1.80, 1.25, 1.00) and lung cancer (rate ratios: Blacks=2.91, 2.18, 1,49, 1.00; Whites=2.08, 2.05, 1.35, 1.00). For women, when differences did exist, the SES gradients were less pronounced for Black women compared with White women. Differences between Black and White women were largest for diabetes (rate ratios: Blacks=1.52, 0.90, 1.27, 1.00; Whites=2.01, 1.22, 1.25, 1.00), COPD (rate ratios: Blacks=1.17, 1.16, 1.14, 1.00; Whites=2.20, 1.45, 1.36, 1.00), and lung cancer (rate ratios: Blacks=1.14, 1.11, 1.36, 1.00; Whites=1.35, 1.05, 1.08, 1.00). For colon cancer, Black women showed a positive SES gradient, whereas White women showed almost no gradient (rate ratios: Blacks=0.72, 0.82, 1.03, 1.00; Whites=0.97, 0.89, 1.03, 1.00).

DISCUSSION

We found markedly higher mortality among lower-SES groups for all specific causes examined, except for colorectal cancer and breast cancer, among employed people aged 35–64 years. Deaths in this age group can all be described as premature and therefore susceptible to intervention. If the mortality rates in the lowest 3 SES quartiles were equivalent to those in the highest quartile (observed rate ratio=1.49), 27% of deaths in this population would have been avoided.

The relatively weak inverse gradient for all-cause mortality for women is partly explained by the positive gradient for breast cancer, the second most common single cause of death among women aged 35–64 years.[21] Excluding breast cancer, the rate ratio for all causes for the lowest SES quartile versus the highest quartile for women increased from 1.29 to 1.38.

Occupation may be less of a valid indicator of SES for women than for men, a circumstance causing misclassification that tends to diminish observed SES gradients. For example, the SES of married women may in some instances be better classified by that of their husbands' occupation rather than their own.[22] Conversely, exclusion of housewives (necessary in our data because we could not assign an occupationally based SES score to housewives) might be expected to improve the accuracy of SES classification for women based on a woman's own occupation. Few data exist with regard to the relative SES gradients for housewives versus employed women, but a Finnish study indicated that whereas SES gradients for employed women were similar to those for men, SES gradients for housewives were weaker than those for men.[21] We checked the validity of our SES variable for women (i.e., the Nam–Powers scores assigned to different occupations) by use of death certificates for the period 1989–1997 that had education data. The mean Nam–Powers scores for male decedents by level of education (less than high school, some high school, high school graduate, some college, college graduate) were 37, 40, 48, 59, and 76, respectively, whereas scores for women were 24, 29, 42, 57, and 74. These scores suggest a reasonable correlation between our SES variable and attained education, which in turn suggests that the SES of women was not greatly more misclassified than that of men. Regardless of the above considerations, results for women in our study may be biased in an unknown manner by the exclusion of housewives.

Strong inverse gradients were found for men for CHD, stroke, COPD, lung cancer, and diabetes; the SES gradients for women for these diseases were weaker but still important. These diseases have risk factors (blood pressure, smoking, and body mass index) that are known to have worse profiles among those with lower SES.[21] These less healthy risk factor profiles are likely to explain some of the observed SES gradients. We had no data on these risk factors and hence were unable to determine whether SES effects persisted independently of these variables.

Breast cancer was the only cause with a markedly positive SES gradient. A positive gradient is typical of breast cancer incidence[23] and is also seen in breast cancer mortality.[10] Much of this gradient is known to be caused by having fewer children and a later age at birth of first child among more educated women, both of which increase breast cancer risk. Adjustment for reproductive risk factors usually reduces the positive SES gradient in mortality,[10] despite the fact that low-SES groups experience decreased survival after diagnosis.[24]

Four other US studies[1,2,8,10] have shown that adjustment for conventional risk factors diminishes but does not eliminate SES gradients for all-cause mortality, CHD mortality, and lung cancer mortality. This finding suggests that (1) unknown or less well-established risk factors associated with SES play an important role in these diseases, (2) treatment affecting survival varies in important ways by SES, or (3) measurement of the risk factors suffers from important errors; probably all 3 possibilities hold. As for less well-established risk factors, Marmot et al.[25] have proposed that job strain may play an important role in heart disease mortality and could explain a large part of the SES effect. However, job strain is usually highly correlated with SES, and researchers have had difficulty separating the effects of job strain from those of SES. To date, there is no consensus on the relative importance of these 2 correlated factors. Two other less well-established risk factors for heart disease that are known to be associated with lower SES are activated factor VII[26] and infection with *Helicobacter pylori*.[27] Homocysteine and C-reactive protein are emerging as important new risk factors for heart disease,[28] but to date we have little information on whether these factors are related to SES.

Whereas in most cases similar SES gradients were seen for both Blacks and Whites, Black men tended to have somewhat steeper SES gradients than White men for most outcomes, especially for lung cancer and stroke. The reverse was true for Black women versus White women, especially for diabetes, lung cancer, and COPD. Obesity differences by SES were less pronounced among Black women compared with White women as of the 1990s,[21] which might account for the smaller SES gradient among Black versus White women for diabetes. In regard to smoking habits, in NHANES III data[20] for adults aged 20–65 years, the percentages of current smokers by descending quartiles of a poverty index were 45, 40, 33, and 23 (White men); 52, 38, 38, and 21 (Black men); 41, 32, 26, and 18 (White women); and 38, 24, 26, and 21 (Black women). The greater differences in smoking prevalence by SES for Black versus White men and the smaller differences for Black versus White women may partly explain differences in SES gradients between Blacks and Whites for lung cancer and COPD. With regard to stroke, unmedicated hypertension among Black men of low SES was higher than that among Black men of high SES in the mid-1990s,[21] whereas the reverse pattern was seen for White men; this difference may contribute to the steeper SES gradient for stroke for Black versus White men.

Our data show a 13% decrease in all-cause mortality for men during the period 1984–1997 (0.9% per year), with the largest increase for the highest-SES group (32%; 1.9% per year). For women, there was a small (6%) decrease, not differentially distributed by SES group. These findings corroborate earlier data.[6,8]

Economic inequality continued to increase during our study period. In 1985, households in the lowest 20% of family income earned 4.0% of the national income, whereas households in the upper 20% earned 45.3%. In 1997, the comparable figures were 3.6% and 49.4%.[29] If we assume that the effects of SES on mortality are causal, increasing inequality (increasing SES differences in the population) would be expected to result in increasing differences in mortality rates between SES groups over time (independent of any possible additional ecological effect of increasing inequality).

We found a 35% decrease in mortality from CHD in the period 1984–1997, with a more rapid decrease for those in higher-SES groups (45% for the highest quartile), especially for men. Similar trends for CHD have been seen in earlier periods.[7–9] The decrease in CHD mortality over time is presumably owing to a combination of improved risk factor profiles and improved treatment. In recent years in the United States, CHD incidence rates (which depend on risk factors) have been stable or have decreased only slightly, whereas mortality rates have continued to decrease sharply[30–33]; these rate patterns suggest that decreasing mortality may be primarily owing to improved treatment. A recent analysis of CHD incidence, mortality, and treatment in 4 US communities led to the conclusion that improved mortality stemmed more from improved treatment than from lower incidence rates.[34] Our own data show that the decrease in CHD mortality was stronger for in-hospital than for out-of-hospital deaths, which also suggests that the overall decline in mortality is driven by treatment factors.

Lower income is related both to access to medical care and to quality of care. In 1998, data from the National Health Interview Survey[35] of adults aged 35–64 years showed that among those below the 21st income percentile, 34% had no medical insurance; among a second group with income ranging from the 22nd to 44th percentiles, 12% had no health insurance; and among those with income above the 44th percentile, 4% had no health insurance (John Pleiss; National Center for Health Statistics, Centers for Disease Control and Prevention; unpublished data; 2001). In the United States, several studies have shown that survival rates and treatment after myocardial infarction are lower for those with lower SES.[36–38] Even in Canada, where there is relatively equal access to medical care, patients' income was strongly related to optimal treatment and survival.[39]

Definitive conclusions about the relative importance of risk factors versus treatment in efforts to explain SES differences in CHD trends would require data on incidence stratified by social class; we lack this data in the United States. Without such data, one cannot rule out that trends in incidence rates may differ by social class in a way that parallels mortality rates as occurs, for example, in Finland.[40]

Lung cancer mortality and COPD mortality also revealed striking SES-related patterns over time in our data. Lung cancer and COPD have been decreasing in men, especially among high-SES groups, whereas they have been increasing in women, especially among low-SES groups. These patterns reflect smoking changes 20–40 years earlier, as more women began smoking and more men quit. Since the 1960s, men and women of higher SES quit smoking at higher rates than did those of lower SES,[22] largely accounting for the SES gradient for these diseases.

In conclusion, rates for the most common causes of death, except for breast cancer and colorectal cancer among women, are higher for lower-SES groups. Better treatment for higher-SES groups may be an important factor in the SES gradient for heart disease, whereas blood pressure, obesity, smoking, and reproductive differences by SES are probably the most important factors that explain the SES gradients for stroke, diabetes, lung cancer/COPD, and breast cancer, respectively. Mortality differences between socioeconomic groups have continued to increase in recent years, particularly for men as income inequality in the United States also increased. ■

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Contributors

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Human Participant Protection

This study was record-based with no personal identifiers and thus was exempt from human subject clearance.

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